U.S. Department of Labor

Office of Administrative Law Judges
2 Executive Campus, Suite 450
Cherry Hill, NJ 08002

(856) 486-3800
(856) 486-3806 (FAX)

Issue Date: 24 March 2006

CASE No.: 2004-BLA-05576

In the Matter of

LONA VARNEY, surviving spouse of
WILLIE G. VARNEY
Claimant

v.

EASTERN COAL CORPORATION
Employer

THE PITTSON COMPANY
c/o ACORDIA EMPLOYERS SERVICE
Carrier

and

DIRECTOR, OFFICE OF WORKERS'
COMPENSATION PROGRAMS
Party-in-Interest

Appearances:

Joseph E. Wolfe, Esquire,
For Claimant

Lois A. Kitts, Esquire,
for Employer/Carrier

Before: Janice K. Bullard
Administrative Law Judge

DECISION AND ORDER
AWARDING BENEFITS

This proceeding arises from a claim for benefits under the Black Lung Benefits Act, 30 U.S.C. §§ 901-945 ("the Act") and the regulations issued thereunder, which are found in Title 20 of the Code of Federal Regulations. Regulations referred to herein are contained in that Title.

Benefits under the Act are awarded to coal miners who are totally disabled within the meaning of the Act due to pneumoconiosis, or to the survivors of coal miners whose death was due to pneumoconiosis. Pneumoconiosis, commonly known as black lung, is a dust disease of the lungs resulting from coal dust inhalation.

Shortly after I was assigned this case, I scheduled it for hearing by Notice issued February 9, 2005. A hearing was held in Pikeville, Kentucky, on May 11, 2005, at which time the parties appeared and presented evidence. I held the record open post-hearing for the admission of additional evidence, which was submitted and admitted to the record. By Order issued September 19, 2005, I closed the record and scheduled the time for submission of written closing argument. Both parties submitted briefs. This decision[1] is based upon an analysis of the record, the arguments of the parties and the applicable law.

I. ISSUE

The sole issue presented for adjudication is whether the Miner's death was due to pneumoconiosis.

II. FINDINGS OF FACT AND CONCLUSIONS OF LAW

A. Procedural Background

On August 30, 2002, Lona Varney ("Claimant", hereinafter) filed the instant claim for survivor's benefits under the Act. DX-3. The District Director of the Office of Workers' Compensation Programs ("Director", hereinafter) determined that Eastern Coal Corp. ("Employer", hereinafter) was the responsible operator for the payment of any benefits due under the Act. DX-18. In its proposed Decision issued September 24, 2003, the Director determined that Claimant was eligible for benefits. DX-26. Employer appealed that finding and requested a formal hearing before the Office of Administrative Law Judges ("OALJ"). DX-27. The matter was referred to OALJ on January 5, 2004. DX-31.

B. Factual Background

Claimant's deceased husband Willie G. Varney ("the Miner", hereinafter) was born on May 22, 1914. DX-1. He married Claimant Lona Mae Goff on April 24, 1945. DX-10. The Miner died on July 8, 2002. DX-11. The Director determined that the Miner had worked in coal mine employment from October 1, 1946 until August 1, 1974. DX-26. This computes to 28 years of coal mine employment.[2] Records relating to the Miner's employment supports this calculation. Employer conceded that it is the responsible operator for payment of any benefits due under this claim. DX-21.

[1] In this Decision and Order, "DX-#" refers to exhibits submitted by Director; "CX-#" refers to exhibits submitted by Claimant; "EX-#" refers to exhibits submitted by Employer/Carrier; "Tr. at #" refers to testimony elicited at the hearing.

[2] Director's DX-26 erroneously cites 23 years of coal mine employment.

The Miner had filed a claim for benefits under the Act during his life, which was denied in a Decision and Order issued by an Administrative Law Judge. That denial was upheld by the Benefits Review Board. DX-1.

At the hearing, Claimant waived testimony. Tr. at 31-32. The parties agreed that survivorship is not an issue in this case. Id.

C. Summary of the Evidence

The claim file developed by the Director contains exhibits identified as DX-1 through DX-33. These were admitted to the record without objection. Tr. at 7. Claimant's exhibit identified as CX-1 was admitted at the hearing, and CX-2 was admitted post-hearing. Employer's exhibits identified as EX-1 through 10 were admitted, but portions of EX-2 were excluded as not timely exchanged, and EX-8 was limited to a discussion of the physician's interpretation of objective tests.

The evidence may be summarized as follows:

1. Death Certificate (DX-11)

A death certificate was issued on August 5, 2002, and was signed by Dr. Tamara Musgrave on July 30, 2002. Dr. Musgrave listed chronic lung disease as the immediate cause of death, and noted aspiration pneumonia and colon cancer as conditions leading to immediate cause of death.

2. Autopsy Report

James A. Dennis, M.D. (DX-12)

Pathologist James A. Dennis performed an autopsy on July 8, 2002. In the report of his findings, Dr. Dennis observed the presence of black pigment deposition in the lungs. The doctor's pathological diagnosis of the respiratory system was:

> 1. Pulmonary congestion mild to moderate with emphysema paniobular and panacinar expressions as well.
> 2. Anthracosilicosis moderate with macule 1.5 cms in focal areas, subpleural in distribution with silical particle impregnation moderate.
> 3. Prominence of pulmonary vessels compatible with cor pulmonale and/or also pulmonary hypertension secondary to vessel manifestations.
> 4. Pulmonary embolus with acute inflammation secondary to the embolic process, no evidence of acute necrosis noted.

Pathological diagnosis of the Miner's cardiovascular system included:

> 1. Left ventricular hypertrophy with hypertensive cardiovascular disease.

2. Coronary artery disease with significant atherosclerotic changes, calcification with focal areas of the left coronary showing greater than 50 to 75% luminal compromise of coronary vessels with extensive calcification and atherosclerotic plague formation.
3. Left ventricular hypertrophy.
4. Remote rheumatic heart disease manifest by chronic inflammatory infiltrate, anascal myelocytes and lipofuschin pigment deposition in association with focal areas of fibrosis in and about or adjacent to the valves with no clinical correlation available.
5. Remote myocardial infarction.

Dr. Dennis provided a summary and discussion of the cause of the Miner's death:

This patient died as a result of cardiovascular disease and coexistant [sic] black lung disease. The pulmonary component of the disease was moderate to severe. Pulmonary hypertension was demonstrated satisfactorily by sections. Anthracosilicosis with macule formation greater than 1 to 1.5 cms and macular changes were demonstrated as well. The pulmonary embolus certainly hastened his problems and was probably secondary to the sedentary changes coexistant [sic] with this disease.

P. Raphael Caffrey, M.D.

Dr. Caffrey reviewed the autopsy slides and report and noted the presence of simple coal workers' pneumoconiosis. DX-15. Dr. Caffrey testified by deposition[3] on November 17, 2003, and reiterated the findings he reported in his written report. EX-7. The doctor is Board certified in anatomical and clinical pathology, and is a Diplomate of the American Board of Pathology. EX-7.

3. Treatment Records

[3] At the hearing, I advised that Dr. Caffrey's opinions would be limited to his observations about the autopsy slides, because any other evidence exceeds the scope of the evidentiary limits set by § 725.414(a)(3)(i). If I were to fully consider his report and testimony, then I would construe Dr. Caffrey's evidence as one of Employer's two permitted medical reports, thereby eliminating from my consideration the evidence from Drs. Rosenberg and Fino, both of whom also testified by deposition, and gave assessments of the Miner's conditions of entitlement. Pursuant to § 725.414(a)(1), "a medical report consists of a physician's written assessment of miner's respiratory or pulmonary condition" and "[a] physician's written assessment of a single objective test, such as a chest X-ray or a pulmonary function test, shall not be considered a medical report." 725.414(a)(1). Dr. Caffrey's interpretation of the autopsy would be admissible, but any other evidence would not. Moreover, his testimony is entirely excludable, pursuant to § 725.414(c), which limits deposition testimony to that from a physician who prepared a medical report or whose testimony is offered in lieu of a medical report. However, as I have considered only that portion of his testimony that corroborates his findings on the autopsy slides, that testimony is merely duplicative of admissible evidence.

DX-12

Reports of June 4, 2001 and June 11, 2001 by Dr. Tamara Musgrave document the Miner's oncology treatment.

DX-13

Report of October 11, 2001 X-ray of the Miner's right foot

Report of X-ray of the Miner's abdomen on October 9, 2001

Reports of laboratory study results

EKG of September 17, 2001

PET scan results form test of August 10, 2001

Report of CT scan of July 11, 2001

Autopsy report duplicated

Report of admission to Pikeville United Methodist Hospital on June 19, 2002, for change in mental status, with final diagnosis of respiratory failure, congestive heart failure, atherosclerotic heart disease, aspiration pneumonitis, coal workers' pneumoconiosis, colon cancer, syndrome of inappropriate antidiuretic hormone secretion, mitral and aortic valve insufficiency, tricuspid valve disease, malnutrition. The report summarized the Miner's treatment during his final hospitalization, ending with his death on July 8, 2002 after increased respiratory compromise.

Report of chest X-ray from September 17, 2001

Report of study of Miner's pulmonary system dated September 18, 2001

Report of X-rays showing colon cancer dated November 21, 2001, November 22, 2001

Reports of chest X-rays of October 8, 2001, October 13, 2001, October 14, 2001, February 13, 2002, May 22, 2002, May 23, 2002, June 19, 2002, June 24, 2002, July 5, 2002, July 8, 2002.

Oncology progress notes of the Miner's treating doctor, Tamar Musgrave, dated April 22, 2002; March 19, 2002; February 12, 2002; January 9, 2002; December 14, 2001; November 19, 2001; August 22, 2001; July 30, 2001.

Report of Barium swallow test of June 26, 2002

Report of test of sinuses of June 26, 2002

CT scan of head of June 19, 2002; May 23, 2002

CT scan of abdomen of May 23, 2002

Report of electroencephalogram of May 23, 2002

Report of chest X-ray of February 17, 1997

DX-14

Report of April 18, 2002 by Dr. Ahmed Malik, noting the Miner's medical history of CAD [coronary artery disease] and pacemaker, as well as angina. The doctor's impression was that the Miner had CAD.

4. Medical Opinion Reports

Joshua A. Perper, M.D. (CX-1; CX-2)

In a report dated February 26, 2003, Dr. Perper summarized his review of the medical evidence of record, including evidence associated with the claim filed by the Miner during his lifetime. CX-1. Dr. Perper wrote that "[t]he U.S. Department of Labor confirmed that Mr. Varney had thirty-nine (39) years of verified coal mine employment." CX-1 at 2. The doctor also noted that Claimant had reported her husband's work in coal mine employment from October 1946 until August 1974. Id. The doctor observed that there was no record that the Miner had smoked. Dr. Perper chronicled the Miner's objective tests and medical history, as well as the treatment records of Dr. Musgrave and records of the Miner's final hospitalization. The death certificate and autopsy report were also summarized.

Dr. Perper found flaws in Dr. Caffrey's review of the autopsy report. The doctor wrote:

> The major flaw of Dr. Caffrey [sic] report is largely his microscopic examination and his interpretation of these findings which clearly and without justification downgrades the severity and significance of Mr. Varney's coal workers' pneumoconiosis and associated pathology.

CX-1 at 12. Dr. Perper disagreed with Dr. Caffrey's report of "only the presence of intra-pulmonary anthracotic macules" and the presence of micronodules only in pulmonary lymph nodes. Id. Dr. Perper asserted that the initial autopsy report and microphotographs that were attached to Dr. Caffrey's report show "evidence of stellate-shaped typical fibroanthracotic micronodules of the mixed coal dust type measuring up to 5 mm." Id. Dr. Perper also faulted Dr. Caffrey's description of the composition of the macules, which he said were of reticulin and anthracotic pigment. Dr. Perper observed that reticulin cannot be "recognized microscopically without special silver" and is "not diagnosable on routine H & E stain (as were stained the autopsy slides of Mr. Varney)." CX-1 at 12. Dr. Perper also concluded that the Miner's emphysema was of severe intensity, and not moderate, as Dr. Caffrey found. In addition, Dr. Perper faulted Dr.

Caffrey for failing to fully discuss the significance of the Miner's emphysema considering his lack of smoking history and his exposure to coal mine dust. Finally, Dr. Perper found that sclerosis of pulmonary vessels was marked, while Dr. Caffrey found it slight[4]. CX-1 at 13.

Dr. Perper examined twenty (20) autopsy slides and observed that "the pleura shows marked, focal pleural and sub-pleural fibroanthracosis with presence of birefringent silica crystals". CX-1 at 13. The slides also revealed sub-pleural areas of nodular fibro-anthracosis, measuring up to 3-4 mm. Throughout the pulmonary parenchyma, the doctor observed macula anthracotic deposits around blood vessels, airways and in the interstitial septa, as well as fibro-anthracotic micronodules of the mixed coal dust type with stellate like shape, surrounded by scar emphysema and measuring up to 5 mm. Moderate to severe centrilobular emphysema was evident and intra-pulmonary blood vessels showed moderate to severe sclerosis. Dr. Perper observed that a peri-bronchial lymph node was largely replaced by silicotic pneumoconiotic nodules with focal necrosis and birefringent silica crystals. A marked, acute and chronic inflammatory infiltrate was present in places in the bronchial walls, with denudation of most of the lining epithelium and a few remnants of metaplastic bronchial epithelia. Small foci of acute bronchopneumonia with the alveoli contained acute inflammatory cells, and an extra-parenchymal pulmonary artery contained a recent thrombo-embolus.

Dr. Perper's review of coronary arteries and the heart showed marked coronary arteriosclerosis and areas of myocardial ischemia. Some of those areas showed physaliherous changes, which the doctor described as vegetal-like cell transformation of myocardial fibers. "Areas of myocardial scarring with entrapped small islands of myocardial fibers" were also noted. CX-1 at 14.

Dr. Perper made the following microscopic diagnoses: 1. Coal workers' pneumoconiosis, simple, slight to moderate severity. 2. Centrilobular emphysema, moderate to severe. 3. Sclerosis of intrapulmonary blood vessels, moderate to severe, consistent with pulmonary hypertension and cor pulmonale. 4. Acute and chronic bronchitis, moderate to severe. 5. Foci of acute bronchopneumonia. 6. Thromboembolus, recent, in extra-parenchymal pulmonary artery. 7. Coronary arteriosclerosis, moderate to marked. 8. Myocardial infarction, remote. CX-1 at 14.

Dr. Perper concluded that "objective evidence indicates that Mr. Varney had significant coal workers' pneumoconiosis", based upon his more than 39 years in underground coal mining; the "presence of worsening chronic obstructive lung disease and mild restrictive lung disease, with marked shortness of breath, decreased breath sounds, abnormal breathing and wheezing, abnormal pulmonary function studies, and slight hypoxemia that required administration of bronchodilators and nebulizers"; progressive respiratory deterioration; autopsy findings showing pneumoconiosis; clinical diagnoses of coal workers' pneumoconiosis; evidence of mild to moderately severe pneumoconiosis with emphysema in a non-smoker; silicotic nodules, which indicated heavy exposure to mixed coal dust containing silica. CX-1 at 17-18.

[4] Dr. Perper's report refers to Dr. Caffrey's findings as "slides", which he corrected to "slight" at his deposition. CX-2 at 17.

Dr. Perper explained the relationship between COPD-centrilobular emphysema and pneumoconiosis where there is a history of exposure to coal dust containing silica and no smoking history. The doctor wrote that "the causal connection between exposure to coal and silica in regard to emphysema and chronic obstructive lung disease is also widely and virtually universally accepted." CX-1 at 18. Dr. Perper also discussed literature that reported the progression of pneumoconiosis after exposure to dust is terminated. CX-1 at 19-20.

In Dr. Perper's opinion, based on the clinical, radiological and laboratory findings, pneumoconiosis was a contributing cause of the Miner's death, and a hastening factor of his death. The doctor concluded that pneumoconiosis contributed to "pulmonary insufficient by direct and extensive replacement of normal lung tissue by non breathing pneumoconiotic lesions and associated centrilobular chronic emphysema, cor pulmonale and resulting hypoxemia, which was also demonstrated clinically". CX-1 at 20. Dr. Perper associated the pneumoconiosis with "hypoxemia precipitating/aggravating a cardiac arrhythmia" and "terminal bronchopneumonia and complicated pulmonary embolus". CX-1 at 20-21.

On July 11, 2005, Dr. Perper testified by deposition, and stated that he has been a practicing physician for about fifty (50) years and is board certified in anatomical, surgical and forensic pathology. CX-2 at 4. The doctor described his experience with pneumoconiosis, and related the factors that must be evaluated in making a diagnosis of the presence of the disease. Id. at 5-6. Dr. Perper testified that he viewed 20 slides from tissue taken from the Miner at autopsy, of which 12 were lung sections. The other slides were sections of the lymph node, pulmonary artery, a section of the aorta, section of the coronary artery of the heart. CX-2 at 10. The doctor observed:

> The lungs show typical characteristic lesions of coal workers' pneumoconiosis, which consisted of the following: the cavity of the lungs, the pleura showed marked focal scarring and thickening with presence of birefringent silica crystals, which indicates again exposure to silica containing coal dust. There were also scattered through the lung tissue, there were nodular anthracotic deposits…which were present around blood vessels, airways and also in the interstitial septa, in the walls between the alveoli. The polarized light again [showed] small birefringent silica crystals…In addition to that, in different places both beneath the pleura, beneath the covering of the lung and also deep and deeper within the lung tissue itself there were what we call nodules of coal workers' pneumoconiosis…

CX-2 at 10-11.

Dr. Perper explained the difference between macro and micronodules, and noted that he observed micronodules in the Miner's tissue, as well as focal and centrilobular emphysema. CX-2 at 12-13. Dr. Perper explained that absent a smoking history, the development of centrilobular emphysema can be attributed to exposure to coal dust, based upon studies that showed the connection between exposure and the disease. CX-2 at 13. The doctor noted the Miner's many years of exposure was consistent with the findings.

Dr. Perper observed small intra-pulmonary blood vessels that showed moderate to serious thickening and scarring or severe sclerosis with marked narrowing of the lumen which is not consistent with hypertension. CX-2 at 14. The doctor also observed that one of the peribronchial lymph nodes was largely replaced by pneumoconiosis nodules, focal necrosis and silica crystals. Id. In addition, the bronchi showed bronchitis and inflammation, and a small area of acute bronchial pneumonia was present.

Dr. Perper testified that the damage to the Miner's lung by silica that he viewed on slides was caused by coal mining because "there was no other reasonable source of silica than the coal dust containing silica." CX-2 at 19-20. Dr. Perper concluded that "the combined severity of the pulmonary damage in the lungs of the miner is a result of the slight moderate coal workers' pneumoconiosis and the complicated centrilobular emphysema was of such severity as to be a substantial configuratory cause of death". CX-2 at 21. Dr. Perper acknowledged that the emphysema would have caused difficulty breathing, which was the reason for his terminal hospitalization. CX-2 at 21. The doctor agreed that the Miner's thromboembolus in a pulmonary artery and pulmonary hypertension were factors that contributed to his death. CX-2 at 21-25.

David M. Rosenberg, M.D.

In a report dated April 6, 2006, Dr. Rosenberg summarized his review of the Miner's treatment records, autopsy report, death certificate, and reports of Drs. Caffrey and Perper. EX-1. Dr. Rosenberg noted the Miner's long history of coal mine employment and his lack of smoking history. The doctor observed that chest X-rays did not reveal micronodularity, but showed changes due to the Miner's other conditions. Dr. Rosenberg cited studies that showed where X-rays did not show pneumoconiosis, pulmonary functions would be expected to be normal, and concluded that the Miner could not be diagnosed with the disease from a clinical point of view. The doctor noted that the minimal degree of pneumoconiosis found by Dr. Caffrey was supported by X-rays that were negative for micronodularity. Dr. Rosenberg concluded that the Miner had a mild degree of pneumoconiosis.

Dr. Rosenberg concluded that the Miner could have performed his coal mine employment, as he "would have been expected to have normal pulmonary functions". EX-1. The doctor observed that a moderate degree of emphysema was noted pathologically, but not on gross inspection of the lungs, and only microscopically. Emphysema also was not apparent on X-rays. Dr. Rosenberg wrote: "it is conceivable the dilation of alveoli seen in Mr. Varney's lungs may have related to the normal aging process". EX-1. The doctor relied upon studies to describe the relationship between emphysema and pneumoconiosis, and concluded that the Miner's emphysema was not related to his coal mine work because the emphysema was not associated with micronodularity.

Dr. Rosenberg concluded that the Miner died of a weakened state with aspiration and pulmonary emboli, and cited articles by other doctors that opined that malignancy is associated with development of pulmonary emboli. The doctor noted that the Miner's advanced metastatic colon cancer was associated with poor prognosis, and because of its advanced stage, "he would have died, as he did in a similar fashion, irrespective of his past coal mine employment." EX-1

at 5. Dr. Rosenberg further concluded "that there is no evidence…that Mr. Varney developed progressive CWP [pneumoconiosis] either of the medical or legal variety after leaving his coal mine employment". In support of that conclusion, the doctor cited treatises and the results of studies that addressed the progressive nature of pneumoconiosis, and which, the doctor concluded, show that simple CWP is not latent and progressive. Dr. Rosenberg also discussed studies and authorities that addressed the issue of progressive and latent chronic obstructive pulmonary disease after cessation of coal mine exposure. Dr. Rosenberg concluded "with respect to the issue of progressive and latent chronic obstructive pulmonary disease in relationship to coal mine dust exposure, there is no scientific foundation…that this occurs." EX-1 at 10. Referring to this conclusion, Dr. Rosenberg opined that the Miner had:

> not developed progressive or latent medical or legal CWP after his coal mine dust exposure ceased. Any simple CWP he had pathologically was not diagnosable on X-ray; also there is no evidence that progression occurred over time, and clearly he did not develop progressive massive fibrosis…The simple CWP he had was just a pathologic finding without adverse physiologic consequences or X-ray correlation. In addition, regarding COPD, there is no evidence this progressed in Mr. Varney; his pathologic finding of emphysema was an incidental finding, unrelated to past coal dust exposure.

EX-1 at 10. Dr. Rosenberg concluded that the degree of pneumoconiosis that the Miner had "would not be expected to be associated with his impairments or disability. Also, he did not have progressive and latent CWP, and his death was totally unrelated to past coal dust exposure. Rather, it related to a weakened state from his metastatic colon cancer with aspiration and pulmonary emboli." EX-1 at 10-11.

On Wednesday, April 20, 2005, Dr. Rosenberg testified by deposition. EX-3. The doctor stated that he is a B-reader and pulmonary and occupational medicine specialist. EX-3 at 19; 3. Dr. Rosenberg discussed his review of the Miner's medical records, including medical reports, arterial blood gas studies, pulmonary function tests, and B-readings of X-rays spanning the years from 1971 through 1994. EX-3 at 20 – 31. Dr. Rosenberg summarized the Miner's medical history as positive for congestive heart failure, with pacemaker and colon cancer and observed that he was in a weakened situation and aspiration. The doctor considered the Miner as "essentially a non-smoker throughout his lifetime." EX-3 at 32. The Miner's respiratory problems were related to congestive heart failure, metastatic carcinoma, aspiration pneumonia, and pulmonary emboli. EX-3 at 32-33. From a clinical perspective, the Miner did not have pneumoconiosis, but the doctor acknowledged that he had a degree of simple CWP. EX-3 at 34.

Dr. Rosenberg agreed that Dr. Dennis' description of the antrasilicosis he found on autopsy was consistent with progressive massive fibrosis, but noted that Dr. Caffrey found that less than 5% of the lung was involved with a mild degree of CWP. EX-3 at 35-36. According to the doctor's review of the records, the only valid pulmonary function tests were performed in 1980 and were normal, and based on that data, the Miner was not disabled from a respiratory standpoint related to pneumoconiosis. EX-3 at 37-39. Dr. Rosenberg denied that the Miner's

death was caused by, related to, or hastened by coal dust exposure, and observed that he would have died from colon cancer regardless of CWP. EX-3 at 39. The doctor found no clear evidence that the Miner had cor pulmonale, and considering that the X-ray evidence was negative for pneumoconiosis, the Miner would not have had functional impairment related to CWP, even though pathologic findings show the presence of the disease. EX-3 at 39-40. The doctor denied that the Miner's cancer would have been hastened by coal dust exposure. EX-3 at 41.

Although it was pointed out to Dr. Rosenberg that the test results he looked at were produced twenty years before the Miner's death, the doctor's opinion remained unchanged. EX-3 at 44. In addition he was asked whether X-rays that were interpreted as positive for pneumoconiosis showed progression of profusion of opacities, but the doctor pointed out that the overwhelming number of X-ray evaluations from May, 1993 onward were negative for the disease. EX-3 at 44-45. The doctor acknowledged that some X-ray readers found evidence of the disease over the years, but he relied upon the preponderance of readings, which were negative. The doctor did not think that those negative readings were called into question by the pathologic findings. EX-3 at 45-46. With respect to the fact that the Miner had emphysema but had not smoked, Dr. Rosenberg discounted exposure to coal dust as the cause of that disease. EX-3 at 48. Dr. Rosenberg agreed that the Miner "probably" had simple pneumoconiosis when he ceased working in the mines, but the presence of coal macules would not have caused functional impairment absent the development of marked scar tissue. EX-3 at 51-52.

Gregory Fino, M.D.

In a report dated April 18, 2005, Dr. Fino referred to his previous report of May 19, 1993, and the records he reviewed at that time. EX-4. Dr. Fino summarized the records he reviewed then, and added a description of updated records that he reviewed to produce the opinions contained in his most recent report. Employer's submission of this report consists of only 2 pages of 10 in which the doctor summarizes evidence he reviewed, and which contains no opinion. This is cured by the attachment of the full report to Dr. Fino's subsequent testimony at deposition at May 2, 2005. EX-5.

In his report of April 18, 2005, Dr. Fino concluded that "it is reasonable to assume that simple coal workers' pneumoconiosis was present based on the autopsy pathology", and further that it was reasonable to assume that the centrilobular emphysema seen on the autopsy "may very well have been related to coal mine dust". EX-5. Dr. Fino found that the panobular or panacinar abnormalities seen on the autopsy were not related to coal dust inhalation. Id. Dr. Fino found it "unreasonable to assume that the pulmonary abnormalities seen pathologically affected [the Miner's] respiratory function either by causing a lifetime impairment and disability or by hastening [his] death", and noted that twenty years after he stopped coal mine employment, he had normal lung function and normal blood gas. EX-5. Dr. Fino concluded that "it is very possible to have coal workers' pneumoconiosis and emphysema seen pathologically without any evidence of respiratory impairment." Id.

Dr. Fino opined that the Miner "died because he was 88 years old and had unresectable lung cancer and severe coronary artery disease. If you will, he died of natural causes. There is

absolutely no evidence whatsoever that any type of lung disease, especially lung disease due to coal mine dust inhalation, played a role in his death". EX-5.

Dr. Fino testified that the Miner's pneumoconiosis was mild to moderate, based upon the opinions of three pathologists. EX-5 at 6. The doctor also noted that two pathologists thought there was evidence of cor pulmonale, and pathologists also found the presence of emphysema. Id. Dr. Fino criticized the autopsy for its limitation to the Miner's lungs and heart, noting that he had colon cancer. Dr. Fino observed that "without a full autopsy, you have no idea whether or not there was an abdominal issue that resulted in his death". Id. at 7. Dr. Fino reviewed clinical records that showed normal pulmonary functioning twenty years after the Miner stopped working, which he concluded demonstrates that the pneumoconiosis present in his lungs did not result in a clinical abnormality. EX-5 at 8. Dr. Fino observed that absent further exposure to coal dust, one would not expect the Miner's condition to worsen between 1993 and his death in 2002. EX-5 at 8.

Dr. Fino did not think smoking played a role in the Miner's death, as he had no real smoking history, but the doctor believed that his colon cancer affected him so as to increase congestion in his respiratory tract, as evidenced by aspiration pneumonia. EX-5 at 8-9. Dr. Fino also noted a history of blood clots in the Miner's lung, which he attributed to his cancer. Dr. Fino denied any relation between inhalation of coal mine dust and the Miner's metastatic colon cancer and coronary artery disease, and concluded that the Miner's simple coal workers' pneumoconiosis did not cause, aggravate or hasten his death. EX-5 at 10.

Dr. Fino assumed that the Miner's emphysema was caused by coal mine dust inhalation, given his lack of smoking history. EX-5 at 11. The doctor conceded that the portions of the Miner's lungs that were involved with simple pneumoconiosis and emphysema would not have functioned properly, but observed that lungs can continue to function because redundancy is built into their structure. EX-5 at 12. Dr. Fino stated that:

> there are numerous studies that look at…operative risks based on pulmonary function testing, and if you have no impairment on pulmonary function studies, and that still could mean you have some disease in your lungs that just had not clinically caused any functional impairment, your risk of a complication or death, as long as your lung function is normal, regardless of whether you have some disease in there or not, your risk is a normal risk.

EX-5 at 16.

5. Other Evidence

The Curriculum Vitae of Dr. Joshua A. Perper is found at CX-1.

Medical articles in support of studies cited by Dr. Rosenberg are included in the record at EX-2.

The Curriculum Vitae of Dr. David M. Rosenberg is found at EX-8.

The Curriculum Vitae of Dr. Gregory J. Fino is found at EX-9.

The Curriculum Vitae of Dr. Raphael Caffrey is found at EX-10.

D. Entitlement

Because this claim was filed after the enactment of the Part 718 regulations, Claimant's entitlement to benefits will be evaluated under Part 718 standards. § 718.2. Section 718.205(a) provides that in order to establish entitlement to survivor's benefits under Part 718, Claimant must prove that the miner had pneumoconiosis, that it arose out of his coal mine employment, and that the miner's death was due to pneumoconiosis. Claimant has the burden of establishing each element of entitlement by a preponderance of the evidence. Director, OWCP v. Greenwich Collieries, 512 U.S. 267 (1994).

1. Pneumoconiosis arose out of coal mine employment

Employer has stipulated that the record establishes that the Miner had pneumoconiosis. Tr. at 32. I find that the record supports this stipulation, as the autopsy report and physician opinion evidence is undisputed on this issue.

A miner who is suffering or suffered from pneumoconiosis and was employed for ten years or more in one or more coal mines is entitled to a rebuttable presumption that the pneumoconiosis arose out of such employment § 718.203(b). Claimant is entitled to this presumption, as the record reflects that the Miner had pneumoconiosis and had worked in coal mine employment for at least twenty-three years. Employer has presented no evidence to rebut the presumption imposed by § 718.203(b), and therefore, I find that Claimant has established that the Miner's pneumoconiosis arose out of coal mine employment.

2. Death due to pneumoconiosis

Death due to pneumoconiosis may be established under § 728.205(c) by any one of the following criteria:

1. Competent medical evidence establishes that pneumoconiosis was the cause of the miner's death.
2. Evidence that pneumoconiosis was a substantially contributing cause or factor leading to the miner's death, or that death was caused by complications of pneumoconiosis.
3. Under § 718.304, the miner suffered from a chronic dust disease of the lung and chest X-ray evidence shows one or more large opacities (greater than 1 centimeter), biopsy or autopsy shows massive lesions in the lung, or other evidence (in accord with acceptable medical procedures) show a condition which could reasonably be expected to yield such large opacities or massive legions.

Section 718.205(c)(5) provides that pneumoconiosis is a "substantially contributing cause" of a miner's death if it hastens the miner's death. § 718.205(c)(5). The medical opinion evidence is in agreement that the Miner had pneumoconiosis.

The record contains no evidence of large opacities, massive lesions, or any other condition which a physician has stated could be expected to result in these. Therefore, the presumptions set forth under § 718.304 are inapplicable here.

Dr. Tamara Musgrave, an oncologist who treated the Miner, was the doctor who issued the Miner's death certificate. Dr. Musgrave listed chronic lung disease among the immediate causes of the Miner's death. As she treated the Miner during his life, I find that her conclusions are supported, and I therefore accord weight to the death certificate. *Dillon v. Peabody Coal Co.*, 11 B.L.R. 1-113 (1988). In assessing this evidence, I have considered the nature of the physician-patient relationship and its duration, and the frequency and extent of treatment. 20 C.F.R. section 718.104(d)(1)-(4). As the miner's treating physician, absolute deference need not be given a treating physician, particularly where there is conflicting evidence. See, Lango v. Director, OWCP, 104 F.3d 573, 577, 21 BLR 2-12 (3d Cir. 1997). The status of treating physician is but one factor to consider; and an administrative law judge must determine whether an opinion is well reasoned. Tedesco v. Director, OWCP, 18 BLR 1-103, 105; 106 (1994). In the instant matter, I find that the record supports according weight to Dr. Musgrave's opinion on the death certificate, as treating records demonstrate that she had a significant relationship with the Miner as his treating physician. I decline to accord controlling weight to Dr. Musgrave's opinion, as she did not provide a full rationale for her conclusions.

Opinions of medical experts are in evidence, including the pathologist who performed an autopsy on the Miner, James A. Dennis, M.D. Dr. Dennis observed the presence of anthracosilicosis and macular changes and concluded that the Miner had pulmonary disease, which he identified as black lung disease. Dr. Dennis found that the Miner died because of cardiovascular disease and black lung disease. I accord weight to Dr. Dennis' opinion, because it is supported by his objective findings, as well as treating records, which show that pneumoconiosis was identified as one of the Miner's diagnoses. An opinion is well-documented and reasoned when it is based on evidence such as physical examinations, symptoms, and other adequate data that support the physician's conclusions. See Fields v. Island Creek Coal Co., 10 B.L.R. 1-19 (1987); Hess v. Clinchfield Coal Co., 7 B.L.R. 1-295 (1984). A medical opinion that is undocumented or unreasoned may be given little or no weight. Clark v. Karst-Robbins Coal Co., 12 B.L.R. 1-149 (1989); see also Duke v. Director, OWCP, 6 B.L.R. 1-673 (1983) (a report is properly discredited where the physician does not explain how the underlying documentation supports his or her diagnosis). Dr. Dennis' opinion is also supported by the Miner's treating physician, which lends it additional weight.

Pathologist Joshua A. Perper, M.D., reviewed the autopsy slides and Dr. Dennis' report and concluded that they showed that the Miner had simple coal workers' pneumoconiosis of slight to moderate severity, as well as emphysema, pulmonary hypertension and cor pulmonale. CX-1. Dr. Perper found the pneumoconiosis "significant", and sufficient to have caused worsening obstructive and mild restrictive lung disease, particularly considering that the Miner had no real smoking history. Dr. Perper opined that the Miner's pneumoconiosis contributed to his pulmonary insufficiency, and stated that the pulmonary damage in the Miner's lungs substantially contributed to the Miner's death. CX-1 and 2. I find this opinion to be well-reasoned and documented, and I accord it substantial weight.

Dr. Rosenberg reviewed the medical evidence of record and observed that chest X-rays did not show micronodularity. Dr. Rosenberg agreed with Dr. Caffrey's findings of minimal pneumoconiosis, based upon the lack of micronodularity in X-rays that he reviewed. Although the doctor acknowledged that some X-rays of record were interpreted as positive for the disease, he relied upon the majority of evaluations that were negative. His conclusion was not affected by the pathology findings, as Dr. Rosenberg concluded that the Miner did not have clinical pneumoconiosis. Dr. Rosenberg also discounted the effect of the Miner's emphysema on his pulmonary function, and concluded that the evidence showed that the miner could have performed his coal mine employment. Dr. Rosenberg reached this conclusion despite acknowledging that the pathology evidence showed emphysema present. Dr. Rosenberg noted that emphysema was not apparent on X-rays and was not noted on gross inspection of the lungs. Dr. Rosenberg conjectured that the dilation of alveoli seen in Mr. Varney's lungs was related to the aging process, and he denied exposure to coal dust as the cause of emphysema. Dr. Rosenberg discounted any relation between the Miner's coal mine work and emphysema, relying upon studies that he believed supported such a finding where micronodularity was not associated with emphysema. The doctor relied upon other studies to support his conclusion that the Miner had "not developed progressive or latent medical or legal [pneumoconiosis] after his coal mine dust exposure ceased", and he found that the degree of pneumoconiosis that the Miner did have would not have caused impairment or disability, and was not related to his death at all. Dr. Rosenberg attributed the Miner's death to his metastatic colon cancer with aspiration and pulmonary emboli.

I find that Dr. Rosenberg's opinion is not well-reasoned. Despite the pathological evidence, Dr. Rosenberg continued to rely upon objective evidence that was produced a decade before the Miner's death. Dr. Rosenberg's explanations and conclusions relied more heavily upon treatises and studies than the evidence of record. Dr. Rosenberg acknowledges the pathological evidence of emphysema, and relied upon a study to find no relation between the Miner's coal mine employment and that disease. The doctor acknowledges no history of smoking, but offers only the conjecture that the Miner's emphysema was possibly related to aging. Dr. Rosenberg found no evidence that the Miner's pneumoconiosis caused any physiologic effects, and he did not address the conclusions of the prosector, or the Miner's doctor, who both found that the disease contributed to his death. I accord little weight to Dr. Rosenberg's opinion.

Dr. Fino's opinions are better reasoned and supported by the record, as the doctor concluded that despite the pathological presence of pneumoconiosis and emphysema, the objective evidence demonstrated that twenty years after the Miner stopped working, he had normal pulmonary function. The doctor believed that absent further exposure to coal dust, the Miner's condition would not have worsened to a disabling degree in the decade between the last objective tests of record and his death. Dr. Fino noted that two pathologists had found cor pulmonale, but noted that the autopsy was limited in nature, and was not complete enough to give a full picture of how his colon cancer may have specifically led to his death. EX-5 at 7. Dr. Fino believed that in the absence of a smoking history, it was reasonable to find that the Miner's emphysema was caused by coal mine dust inhalation, and he observed that despite the evidence showing this disease and pneumoconiosis in the Miner's lungs, an individual could still function normally. The doctor concluded that the Miner's death was due to lung cancer and severe coronary disease, and unrelated to his coal dust inhalation.

I accord weight to Dr. Fino's opinion, as it is supported by the record. However, I am persuaded by the opinions of the Claimant's treating doctor, combined with those of the physician who performed the autopsy, corroborated by Dr. Perper's well-documented and well-reasoned opinion, that the Miner's pneumoconiosis contributed to his death. Dr. Dennis found that the pulmonary embolus that hastened the Miner's death was related to pneumoconiosis. Despite Dr. Fino's contention that the Miner would have died as he did despite the presence of pneumoconiosis because of his age and the extent of his cancer and cardiac disease, Dr. Fino did not offer a well-documented opinion that the Miner would necessarily have developed a pulmonary embolus, absent pneumoconiosis. In contrast, Dr. Perper observed that the Miner had observable damage to his lung that was caused by coal dust, and he further found that the Miner's emphysema would have caused difficulty breathing, which was the reason for his terminal hospitalization.

In consideration of the record as a whole, I find that it supports finding that the Miner had pneumoconiosis that contributed to or hastened his death.

I further assign less weight to the opinions of Drs. Fino and Rosenberg because neither examined the Miner, in contrast to two physicians whose treatment of the Miner is well-documented in the record. *Cole v. East Kentucky Collieries*, 20 B.L.R. 1-51 (1996). Although both doctors are well-qualified pulmonary experts, in consideration of the evidence of record, I find it appropriate to accord more weight to the opinions of the Miner's treating physician, Dr. Musgrave, and the physician who performed his autopsy, Dr. Dennis.

I note that the Miner was credited with fewer years of coal mine employment than was assumed by some of the physicians of record. I note that Dr. Perper referred to the Miner's 39 year history of coal mine employment, but at the same time correctly reported his work as a miner between October 1946 and August 1974, which comports with the 28 years that I have credited as coal mine employment. See, CX-1. I find this an insignificant discrepancy, as all of the physicians assumed a similarly inflated period of coal mine employment, and there was no evidence of record of any other exposure to a lung infiltrate, such as that caused by a significant smoking history.

III. CONCLUSION

The Claimant established that the Miner worked in coal mine employment for at least twenty-three years. In consideration of all of the evidence of record, I find that the evidence establishes the presence of pneumoconiosis, and further supports a finding that the Miner's death was due, at least in part, to pneumoconiosis.

ATTORNEY FEE

No award of attorney's fees for services to Claimant is made herein because no fee application has been received. Thirty (30) days is hereby allowed Claimant's counsel for the submission of a fee application that must conform with §§ 725.365 and 725.366 of the regulations. A service sheet showing that service has been made upon all parties including Claimant must accompany the application. Parties have ten (10) days following receipt of any

such application within which to file any objection. The Act prohibits the charging of a fee in the absence of an approved application.

ORDER

The survivor's claim of LONA VARNEY for benefits under the Act is AWARDED.

A

Janice K. Bullard
Administrative Law Judge

Cherry Hill, New Jersey

NOTICE OF APPEAL RIGHTS: If you are dissatisfied with the administrative law judge's decision, you may file an appeal with the Benefits Review Board ("Board"). To be timely, your appeal must be filed with the Board within thirty (30) days from the date on which the administrative law judge's decision is filed with the district director's office. *See* 20 C.F.R. §§ 725.458 and 725.459. The address of the Board is: Benefits Review Board, U.S. Department of Labor, P.O. Box 37601, Washington, DC 20013-7601. Your appeal is considered filed on the date it is received in the Office of the Clerk of the Board, unless the appeal is sent by mail and the Board determines that the U.S. Postal Service postmark, or other reliable evidence establishing the mailing date, may be used. *See* 20 C.F.R. § 802.207. Once an appeal is filed, all inquiries and correspondence should be directed to the Board.

After receipt of an appeal, the Board will issue a notice to all parties acknowledging receipt of the appeal and advising them as to any further action needed.

At the time you file an appeal with the Board, you must also send a copy of the appeal letter to Allen Feldman, Associate Solicitor, Black Lung and Longshore Legal Services, U.S. Department of Labor, 200 Constitution Ave., NW, Room N-2117, Washington, DC 20210. *See* 20 C.F.R. § 725.481.

If an appeal is not timely filed with the Board, the administrative law judge's decision becomes the final order of the Secretary of Labor pursuant to 20 C.F.R. § 725.479(a).